REMARKS

Favorable reconsideration is respectfully requested in view of the foregoing amendments and the following remarks.

I. CLAIM STATUS & AMENDMENTS

Claims 1-9 were pending in this application when last examined and stand rejected.

Applicants thank Examiner Natarajan for examining all the claims in this application together as noted in items 2-3 on page 2 of the Office Action.

Support for the amendment to claim 7 can be found in the disclosure, example, at page 4, lines 17-22. Kindly note that a copy of the Deposit Receipt of the hybridoma of claim 7 deposited as FERM BP-5631 was submitted with the application filing papers.

New claims 10-13 have been added. Support for new claims 10-11 and 12-13 can be found in the disclosure, example, at page 19, lines 21-24, and page 25, lines 1-25, respectively.

No new matter has been added.

Claims 1-13 are pending upon entry of this amendment.

II. INDEFINITENESS REJECTION

Claim 7 was rejected under 35 U.S.C. § 112, second paragraph, as indefinite for the reasons set forth in item 4 on page 2 of the Office Action.

The amendment overcomes this rejection. In particular, claim 7 is amended to clarify that the humanized antibody is a humanized antibody produced by hybridoma clone deposited as FERM BP-5631.

Therefore, this rejection is untenable and should be withdrawn.

III. WRITTEN DESCRIPTION & ENABLEMENT REJECTIONS

Claims 1-9 were rejected under 35 U.S.C.§ 112, first paragraph, on the basis that the specification lacks written description support for the claimed invention for the reasons set forth in item 5 on pages 3-4 of the Office Action.

In item 6 on pages 4-6 of the Office Action, claims 1-9 were rejected under 35 U.S.C.§ 112, first paragraph, on the basis that the specification is enabled for inducing apoptosis *in vitro* by inhibiting binding of parathyroid hormone related peptide to a receptor thereof, but not for *in vivo* methods involving administering to a subject a substance that induces apoptosis by inhibiting binding of parathyroid hormone related peptide to a receptor thereof to treat chondroma and chonrosarcoma.

Applicants respectfully traverse these rejections.

Starting with the written description rejection, The Office contends that the application does not provide any and all exemplary species of the genus of any substance that inhibits binding of parathyroid hormone related peptide to a receptor thereof. The Office argues that the specification does not identify any characteristics other functional limitations (i.e., inhibiting binding). The Office contends that the application only discloses the possession of an "anti-PTHrP" antibody and not all "substances that inhibit binding.

Applicants respectfully disagree and submit that one of ordinary skill in the art, upon reading the disclosure and in view of the knowledge in the art, would reasonably believe that Applicants were in possession of the invention as claimed.

On pages 5-6, the specification discloses and defines the "substance of the invention that inhibits binding of PTHrP and PTHrP receptor" as either one or both substances that bind to PTHrP and prevents such from binding to a PTHrP receptor. Examples include an anti-PTHrP antibody and antagonists to a PTHrP receptor (i.e., "PTHrP antagonists"). Such PTHrP antagonists include a polypeptide having PTHrP antagonist activity or a low molecule substance. Such PTHrP antagonists include those as described in JP Patent Publication (Kokai) No.

7-165790A (1995), JP Patent Publication (Kohyo) No. 5-509098A (1993), Peptides (UNITED STATES), vol. 16, no. 6, pp. 1031-1037, 1995, Biochemistry (UNITED STATES), vol. 31, no. 16, pp. 4026-4033, April 1992.

The specification even provides a working example of an anti-PTHrP antibody. See pages 6-7 and the Examples on pages 62-64. The specification even discloses making humanized forms of the antibody common and routine techniques in the biotechnology industry. See the disclosure of a humanized antibody produced by hybridoma clone deposited as FERM BP-5631 at page 4, lines 17-22. In addition, the specification discloses making and using antigen-binding fragments of such antibody utilizing common and routine techniques in the biotechnology industry.

Thus, contrary to the Office's assertion, the specification provides a sufficient description of a representative number of species of the claimed invention. In other words, the specification provides full written support for the genus of substances of the claims, such that one of ordinary skill in the art would reasonably believe that Applicants were in possession of the claimed invention at the time of filing of the instant application.

Moreover, as to enablement, the specification demonstrates that such anti-PTHrP antibody induces apoptosis *in vitro* through the control of Bcl-2/Bax and caspase 3. See, for instance, pages 62-64. A person of ordinary skill in art, upon reading this disclosure and in view of the knowledge in the art, would reasonably expect the same *in vivo* effect of apoptosis, based on the *in vitro* effect of apoptosis. Also, a person of ordinary skill in the art would expect that a substance that could be used for treating chondroma and chondroma, especially that associated with apoptosis mediated by Bcl-2/Bax and caspase 3. See for instance, the background of the invention section and Famum et al., Am. J. Anat., vol. 186, pp. 346-358, 1989; Lewinson et al., Anat. Rec., vol. 233, pp. 504-514, 1992, and Amling et al., J. Cell. Biol., vol. 136, pp. 205-213, 1997.

The specification describes the mechanism of the claimed invention. The relationship between apoptosis and inhibition of cancer has been fully confirmed as discussed above.

On page 17, the specification describes methods of administering the agent to treat

chondroma and chondrosarcoma, including how to formulate effective amounts and dosages.

Based on the above, one of ordinary skill in the art could readily identify substances that inhibit binding of parathyroid hormone related peptide to a receptor thereof (claim 1) and use such substances to treat chondroma and chondrosarcoma (claim 1) and/or induce apoptosis in chondroma and chondrosarcoma cells (claim 8) without undue experimentation.

Thus, Applicants respectfully submit that the specification provides full written description and enabling support for the claimed invention and the rejections should be withdrawn.

IV. ANTICIPATION REJECTION

In item 7 on pages 6-7 of the Action, claims 8 and 9 were rejected under 35 U.S.C. § 102(a), as anticipated by Miyaji et al. (The Japanese Cancer Association Sokai Kiji, p. 174, Aug. 25, 2002) (cited in the IDS of May 5, 2005).

The Office has acknowledged the foreign priority claim, as well as receipt of the certified priority document in this application. See item 12(a)1 on page 1 of the Office Action. Accordingly, the instant application can claim priority back to JP 2002-334081, filed November 18, 2002.

Moreover, this rejection is respectfully traversed on the basis that the Miyaji et al. reference is a publication written by the present inventors, and therefore, does not correspond to a "publication" under 35 U.S.C. § 102(a). Applicants submit that the authors other than the inventors did not contribute to the present invention.

Thus, the rejection should be withdrawn.

V. CONCLUSION

In view of the foregoing amendments and remarks, it is respectfully submitted that the present application is in condition for allowance and early notice to that effect is hereby requested.

If the Examiner has any comments or proposals for expediting prosecution, please contact the undersigned attorney at the telephone number below.

Respectfully submitted,

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